ON CERTAIN CHANGES IN THE ELASTIC TISSUE OF THE PELVIS OF THE KIDNEY, AND THEIR RELATION TO HYDRONEPHROSIS.

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(From the Russell Sage Institute of Pathology.)

Inducement to this study was given by a frequent occurrence of a dilated pelvis of the kidney, which assumed at times extreme dimensions in cases of arteriosclerotic and senile kidneys, and in advanced productive nephritis. No extrarenal obstruction or anomalies of insertion or angle of the ureter could be discovered.[1]

In some of these cases the kidneys presented the picture of a long-standing, double hydronephrosis, although the extent of the lesion was at times different on both sides. In a number, particularly the aged, the lesion had become associated with an inflammatory condition of the pelvis, but only in a few it assumed a purulent character.

These observations extended over 505 autopsies performed during the year 1909. Among them were 15 which showed a marked dilatation of the pelvis of the kidney; in 3 this was complicated by a purulent pyelitis. In none was any common obstruction or abnormality in the relationship of the ureter to the pelvis discovered, which would account for retention of urine. The ureter was not dilated in these cases, but the walls of the pelvis of the kidney were considerably thickened. The urethra was found intact, and the walls of the bladder were not affected. In several cases the disease was not the same on both sides, and in some early instances parts of the kidney were still intact, while in others considerable flattening of papillæ had taken place with much distention of the calices. Below are cited the extreme cases:

Case I.—An emaciated man, aged seventy years, had died from lobar pneumonia of the upper and middle lobes of the right lung and pneumococcal meningitis. The heart (510 grams) was considerably hypertrophied, particularly the wall of the left ventricle. Urinary bladder and urethra presented no special change. The renal arteries were both somewhat atheromatous. The renal cortex was fairly well preserved. The right kidney (112 grams) was somewhat reduced in size, its capsule slightly adherent and thickened, its cortex irregularly reduced, and its vascular markings irregular as in the usual forms of arteriosclerotic atrophy of the kidney. The pelvis of this kidney was considerably distended and its walls thickened. The left kidney was virtually transformed into a sac. Its pelvis was enormously distended and the kidney substance almost completely lost. Microscopically, what kidney tissue had remained showed the glomeruli almost completely overgrown with fibrous tissue, and the tubules obliterated. In some remaining tubules the lumen had become largely distended and their lining cells transformed into flattened epithelium.

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[1] An illustration of this disease will be found in my monograph, The Anatomic Histological Processes of Bright's Disease, Philadelphia, 1910, Fig. 45.

Case II.—An emaciated woman, aged eighty-eight years, had been under observation for progressive weakness and mental enfeeblement for six months. Her urine had been increased in amount and always presented a small amount of albumin and casts. Toward her end she became edematous and died of cardiac failure. Autopsy showed a heart of 300 grams, atrophic. There existed general arteriosclerosis. Both kidneys were considerably reduced in size (150 grams); the pelvis in both was markedly dilated and its walls slightly thickened. The urinary bladder was somewhat dilated and its walls thin and atrophic. There was no suppurative condition in any part of the urinary tract.

Case III.—The third case presented much the same appearances, but had a superadded pyelitis. It occurred in a wasted male, aged sixty-nine years, who for one year had been progressively wasting, and had been recognized to have nephritis. There was a slight increase in the amount of urine with some albumin and casts, but never any pus. He died of gradual cardiac insufficiency. At autopsy the heart (300 grams) was atrophic, and there was a considerable degree of arteriosclerosis. The right kidney (150 grams) showed a moderate degree of arteriosclerotic atrophy, and the left (75 grams) was much reduced in size, while on section its pelvis was markedly dilated, and the calyces were so distended as to almost completely obliterate the kidney tissue. The pelvis was filled with a thick cheesy pus. Microscopically, no organisms were found, but the lesion was certainly not tuberculous. The ureters were patent and the urinary bladder was not hypertrophied nor did it contain any purulent material. There was a history of gonorrhea twenty years previously, but no apparent trace of this was determined at autopsy.

We deal, therefore, in this series with cases of hydronephrosis of all grades, from dilated calices to an almost complete obliteration of kidney substance and the formation of a large sac; some complicated by pyelitis; in all the direct cause of the lesion was obscure.

In a recent very extensive experimental and anatomical study of this disease, Ponfick[2] classifies it into two great groups, one with manifest, the other with obscure or doubtful etiology. Of a total number of 76 cases studied, 53 had a manifest cause, while as many as 26 belonged to the latter and most interesting class. It is frequently asserted that hydronephrosis of obscure origin finds an explanation in a valve-like reduplication of a fold of mucous membrane at the junction of ureter with kidney pelvis. Ponfick, however, attributes such changes in the mucous membrane in infantile and in a certain number of cases of adult hydronephrosis to urinary concretions, which, having become lodged at the orifice of the ureter, produce urine stasis, and by irritative processes give rise later to the formation of an obstructing fold of mucous membrane, which persists even after disappearance of the concretion. In Ponfick's opinion the condition is, therefore, acquired and not developmental. There still remain, however, cases of obscure hydronephrosis without any such fold formation. Ponfick believes these due to an abnormal course of the ureter, producing an abnormal degree in the angle which the ureter forms with the pelvis of the kidney. Incidentally Ponfick mentions that the progress of the lesion may be aided by a gradual atrophy of the contractile elements which accompany the induration of the intermuscular connective-tissue bands of the pelvis. It does not appear that he attributes to these anatomical changes in the pelvis any essential role for the production of the hydronephrotic process.

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[2] Beiträge zur pathologischen Anatomie begründet von Ziegler, Band xv, Heft 1.

The explanations of Ponfick are not applicable to the cases under consideration. In none of them were there evidences of previous concretions with changes in the mucous membrane or an abnormal angle or course of the ureter. Moreover, the irregularity in the development, as exemplified by early cases, its constant occurrence in diseases associated with slowly progressing atrophy of renal substance, and in the aged, suggested that pathological changes in the contractile elements might be of greater consequence than usually supposed. For the study of the changes in these structures the kidneys of about 100 cases of various kinds were sectioned.

The normal mucous membrane of the pelvis of the kidney contains a considerable amount of elastic tissue, which is regularly distributed in delicate fibers between the connective tissue of the mucosa, submucosa, and the muscle bundles. Elastic fibers enter, therefore, abundantly and widely into the structure of this mucous membrane. It has been found that this elastic tissue shows marked and important changes in various diseases of the kidney.

In the type of cases related above, that is, in the senile, arteriosclerotic kidney and in productive nephritis, in so far as they are uncomplicated by any diffuse inflammation of the pelvis of the kidney, the elastic tissue had become very prominent and was apparently much increased, while the connective tissue and muscular elements were atrophic and in extreme cases lost. Thus, in certain cases the elastic elements made up almost the entire bulk of the pelvic tissue. These differed, however, very markedly from the delicate, evenly distributed fine fibers found in the normal mucous membrane. They were coarse, in places very thick and hyaline, fibrillated, frequently broken and disintegrated. The increase of elastic elements seems to be particularly marked near places where loss or fibrous contraction of kidney substance has occurred; for instance, immediately adjoining the flattened papillary portions of the kidney substance. The pictures thus obtained remind one very much of the changes occurring in arteriosclerotic arteries.

In all cases of pyelitis, whether from direct obstruction or implanted on a preëxisting hydronephrosis, or complicating a productive nephritis, the elastic tissue is destroyed, in purulent cases very rapidly, and eventually disappears entirely. This change is analogous to the behavior of elastic tissue in other inflamed organs. Non-purulent pyelitis, particularly near the papillary portion in close proximity to the kidney substance, is frequent in productive nephritis. It is difficult to determine its exact relation to the nephritic process, but it has a destructive influence on the elastic tissues of the pelvis. In this event there occurs also a gradual dilatation of calices and kidney pelvis. When this inflammatory process is localized it may be followed by elastic thickening of neighboring parts.

In a case of obstructive hydronephrosis, complicated by a pyelitis due to a cancer of the uterus, there occurred complete loss of elastic tissue. It would be interesting to investigate more thoroughly in the future the behavior of the elastic tissue in uncomplicated obstructive hydronephrosis, as well as the fate of the elastic tissue in specific inflammations of the pelvis of the kidney.

It would appear that the increase in elastic elements in the form of coarse lamellæ is probably a compensatory change incident to primary weakening and disintegration of these structures, together with progressing atrophy of the other component parts of the kidney pelvis. As in the case of arteriosclerotic arteries, it never appears to succeed in fully establishing reparation, but is prone to undergo disintegration. Thus a progressive weakening of these structures occurs, which in association with the atrophy of the fibrous and muscular parts leads to dilatation of the pelvis and stagnation of urine and true hydronephrosis.

It is not easy to determine exactly how far inflammatory conditions within the kidney parenchyma contribute toward these events. They may aid by a reduction of kidney substance, or a co-existing localized inflammatory extension within the pelvis of the kidney may be the factor. In such cases compensatory elastic thickening follows. But it has not been possible to demonstrate the formation of new elastic fibers after they have once been extensively destroyed by a general purulent pyelitis.

It does not seem that an increased urine output is of essential consequence, as other diseases with marked polyuria, but without changes in the elastic tissue of the kidney pelvis, apparently do not lead to hydronephrosis.

Practically it remains important that an idiopathic hydronephrosis, which at times is conspicuously associated with inflammatory changes or even purulent pyelitis, is apt to occur in elderly people, and in those with contracted kidneys.

THROMBO-ANGIITIS OBLITERANS: A CLINICAL AND PATHOLOGICAL STUDY.

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Presenile spontaneous gangrene,[1] or so-called juvenile gangrene, at one time belonged to that ill-defined group of symptom complexes known as the vasomotor trophoneuroses. This group was made to embrace allied or intermediate conditions, including simple acrocyanosis[2] erythromelia, acroparesthesia[3] erythromelalgia,[4] and Raynaud's disease.[5]

From Raynaud, Mitchell, von Winiwarter, Munro, Cassirer, Strauss,[6] and, finally, Buerger, we have had various contributions on the clinical and pathological aspects of one or more of the above-named conditions. An analysis of the clinical signs and symptoms of any one of these conditions will show that they manifest themselves by a combination of one or all of three main symptom groups—vasomotor, sensory, and trophic.[7] The vasomotor include hyperemia, syncope, asphyxia; the sensory, pain, hyperesthesia, and paresthesia; the trophic, ulceration, gangrene, and dystrophies of the skin. Acryocyanosis, the simplest type, represents one symptom from the vasomotor group. Erythromelalgia includes hyperemia from the vasomotor group, and pain from the sensory. Raynaud's disease embraces all three groups; of these the vasomotor are hyperemia, syncope, and asphyxia;

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[1] Leo Buerger, Thrombo-angiitis Obliterans, Amer. Jour. Med. Sci., 1908, cxxxvi, 567.

[2] Cassirer, Die Vasomotorisch tropischen Neurose, S. Karger, Berlin, 1910.

[3] Schultze, Ueber Akroparesthesie, Deutsch. Zeitsch. f. Nervenh., 1892, iii, 300.

[4] S. Weir Mitchell, Phila. Med. Times, November 23, 1872.

[5] M. Raynaud, Thèse de Paris, 1862.

[6] Arch. f. Psychiat., 1905, xxxix, 109.

[7] Barker and Sladen, Jour. Nerv. and Ment. Dis., December, 1907.

the sensory, pain, anesthesia, and paresthesia; the trophic, gangrene.[8]

Unfortunately, the pathological interpretation of these affections has not been clearly elaborated. Clinical classification, however, has advanced more rapidly and we have now numerous, nearly allied clinical conditions, satisfactorily classified, but without adequate pathological explanation. In the attempt to offer a pathological explanation for presenile spontaneous gangrene, von Winiwarter, in 1879, claimed that the terminal gangrene in many of these cases was the result of an occlusion of the lumen of the vessels by means of an obliterating endarteritis, which consisted in proliferation of the fibrous and cellular elements of the intima—endarteritis obliterans. Weiss and von Monteuffel,[9] subsequent to von Winiwarter, claimed that the disease is caused by a primary endarteritis, with the formation of a white descending thrombus, with subsequent organization and arteritis.

That *senile* gangrene is due, in most cases, to endarteritis obliterans is not questioned, but Buerger,[10] in 1908, advanced a new view, maintaining that *presenile* spontaneous gangrene was not a protean manifestation of an arteriosclerotic process, but an altogether new pathological entity, depending upon the primary formation of *obturating thrombi* in the arteries and veins, and to which he gave the name thrombo-angiitis obliterans.[11] He showed that the red thrombus is the precursor of the condition; that the process does *not* extend from above but from below upward; and that the lesion may occur in vessels devoid of endarteritis.

According to Buerger,[12] presenile spontaneous gangrene is a distinct clinical and pathological entity, characterized by thrombotic occlusion of arteries alone, or of arteries and veins, giving subjective manifestations, chief among which are pain and the peculiar symptom of intermittent claudication, and presenting objective phenomena, the most important of which are redness in the dependent position of the limb (erythromelia), marked blanching in the elevated position, evidence of arterial occlusion in the form of pulseless vessels (popliteal, anterior and posterior tibial, dorsalis pedis, etc.), trophic disturbances of moderate extent and of even grave consequence, often terminating in gangrene of one or both lower extremities. Clinically, thrombo-angiitis obliterans may show many variations from a set form, and this accounts for the fact that the true condition is so frequently over-

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[8] Barker and Sladen, loc. cit.

[9] Cited by Buerger and Brooks, Jour. Med. Research, September, 1911, vol. xxv, No. 247.

[10] Loc. cit.

[11] Quite recently Ch. Baumler describes this condition under the caption Arteritis Thrombotica, v. Benzoldt and Stintzing, Med. and Therapie. (Personal communication from Dr. Buerger.)

[12] Amer. Jour. Med. Sci., January, 1910.